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USPT	(APC or antigen adj presenting adj cells) near5 FADD same Fas same apoptosis	0	L1

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L6: Entry 1 of 1 File: USPT Jan 4, 2000

DOCUMENT-IDENTIFIER: US 6010853 A
TITLE: Siva genes, novel genes involved in CD27-mediated apoptosis

DEPR:
The Siva proteins or biologically active portions thereof of the invention can have one or more of the following biological activities: 1) they can interact with (e.g., bind to) CD27, e.g., the cytoplasmic tail of CD27; 2) they can modulate the activity of CD27; and 3) they can modulate or regulate apoptosis, e.g., apoptosis of immune cells. Thus, in a preferred embodiment of the invention, the Siva molecules modulate CD27-mediated apoptosis of cells, e.g., immune cells. As used herein, the language "immune cell" refers to hematopoictic cells such as T lymphocytes, B lymphocytes, monocytes, macrophages, dendritic cells, and other antigen presenting cells, natural killer (NK) cells, and lymphokine activated killer (LAK) cells. Thus, as elevated levels of solible CD27 have been reported in cases of multiple sclerosis (Hinzten, R. Q. et al. (1991) J. Neuroimmunol. 35:211-218), and such elevated levels may inhibit the regulatory or other effects of CD70-expressing and B cells, the Siva molecules (or modulators thereof) of the invention, which can modulate the activity of CD27, can be used to treat multiple sclerosis and other autoimmune diseases. It is also known that B cell cancers such as Non-Hodgkin's lymphoma and B cell chronic lymphocytic leukemia (B-CLL) do not undergo apoptosis despite the high expression of both CD27 and CD70. This is due to the fact that these cells release soluble CD27 which builds up in the body fluids and disrupts CD27-mediated apoptosis. Thus, the Siva molecules (or modulators thereof) of the invention can also be used to treat immune cell proliferative disorders such as Non-Hodgkin's lymphoma and leukemias. Moreover, disruption of binding between CD27 and CD70 by soluble CD27 aids in metastasis by, for example, loosening the homotypic cell--cell contact occurring through CD27 and CD70 interaction. By promoting apoptosis of the tumorigenic cells, the Siva molecules (or modulators thereof) of the invention can be used to inhibit tumor metastasis.

DEPR:
Thus far, two types of molecules that interact with various TNFR family members have surfaced. One group comprises the death domain containing proteins-TRADD, FADD and RIP which interact with Fas and TNFRI (Hsu, H. et al. (1995) Cell 81:505-512; Chinnaiyan, A. M. et al. (1995) Cell 81:505-512; Stanger, Z. B.

et al. (1995) Cell 81:513-523; Hsu, H. et al. (1996) Immunity 4:387-396). TRAFs form the second group, characterized by the zinc finger domains and interact with TNFRII, CD40 and LMP1 (Mosialos, G. et al. (1995) Cell 80:389-399; Rothe, M. et al. (1995) Science 269:1424-1427; Hsu, H et al. (1994) J. Biol. Chem. 269:30069-30072; Cheng, G. et al. (1995) Science 267:1594-1498). In comparison, Siva does not appear to fall into either of these categories. Using the Clustal program that takes into consideration both the size and hydrophobicity of amino acids, a region in Siva homologous to the known death domains of FADD and RIP was identified. Although overall homology between the three is high, identical homology is low. Dendrogram analysis clearly places Siva outside all of the known DD containing proteins. The homologies reported here are comparable to those calculated for TRADD/FADD and TRADD/RIP and are higher than those for the DD of Reaper and other DD containing proteins (Cleveland, J. L. and Ihle, J. N. (1995) Cell 81:479-482). NMR structure of Fas DD revealed the presence of six antiparallel, amphipathic .alpha. helices, and a similar structure has been proposed for other DDs (Huang, B. et al. (1996) Nature 384:638-641). However, based on secondary structure predictions, the DDHR of Siva appears to lack at least 4 of these helices and thus could possibility be structurally different from that of the DD of Fas and its distant relative Reaper. An important consideration is that all DDs do not appear to be similar in terms of cellular function. For example, the DD of FADD is required for binding of FADD to FAS DD, but not for induction of apoptosis (Stanger, Z. B. et al. (1995) Cell 81:513-523; Grimm, S. et al. (1996) PNAS 93:10923-10927). TRADD DD however is required for eliciting both apoptosis and activation of the transcription factor NFkB (Chinnaiyan, A. M. et al. (1995) Cell 81:505-512; Park, A. and Baichwal, R. (1996) J. Biol. Chem. 271:9858-9862). In case of the Drosophila protein Reaper, mutations carried out in the DD region similar to that of Fas DD, does not abrogate the potent apoptotic activity of the protein (Chen, P. et al. (1996) J. Biol Chem. 271:25735-25737).

DEPV:

a) a death domain homology region (DDHR) which appears at amino acid residues 62 to 136 of SEQ ID NO:2 and which is also shown in the present application as a separate sequence designated SEQ ID NO:7. As used herein, a DDHR refers to a region of a Siva protein which includes at least about 40, preferably at least about 50, more preferably at least about 60, still more preferably at least about 70, and most preferably at least about 80 amino acid residues or more and which is at least about 10% or more homologous to the death domain of FADD (Chinnaiyan, A. M. et al. (1995) Cell 81:505-512), RIP (Stanger, B. Z. et al. (1995) Cell 81:513-523), TRADD (Hsu, H. et al. (1995) Cell 81:495-504), or Fas (Cleveland, J. L. and Ihle, J. N. (1995) Cell 81:479-482). The DDHR typically is a region of a protein which is involved in apoptosis. The DDHR can also be involved in, for example, modulation of transcription. In one embodiment, the DDHR of Siva is involved in activation of transcription factors such as NFkB. Methods for measuring NFkB activation are known in the art. Hsu, H. et al. (1995) Cell 81:495-504. In preferred embodiments, a Siva protein of the invention includes a DDHR which is at least

about 50%, more preferably at least about 60%, still more preferably at least about 70%, and most preferably at least about 80, 85, 90% or more homologous to the DDHR of Siva-1 as shown in SEQ ID NO:7.;

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L4: Entry 2 of 41 File: USPT Jun 19, 2001

DOCUMENT-IDENTIFIER: US 6248875 B1
TITLE: Neuronal MORT1 isoforms

BSPR:
Apoptosis, or programmed cell death in multicellular organisms, is one of the fundamental means by which a cell can respond to environmental changes. One of the best studied mammalian apoptosis systems involves Fas (also designated APO-1 and CD95), a type I membrane receptor that, when crosslinked by its cognate ligand, induces apoptosis in a wide variety of cells (for review, see Nagata, 1994). The extracellular interaction of Fas ligand with the cell membrane-spanning Fas receptor activates an intracellular signal transduction cascade finally activating proteases in the IL-1.beta.-converting enzyme (ICE) family (Henkart, 1996). Transduction of an apoptosis signal depends on interaction between the intracellular "death domain" of Fas with a cytoplasmic 23-kDa protein, MORT1 [(Boldin, et al., 1995), also termed FADD (Chinnaiyan, et al., 1995)]. The events leading from the production of an activated Fas trimer complex to cell destruction mediated by ICE-like proteases are yet to be determined, but recruitment of two MORT1/FADD molecules into a death-inducing signaling complex with the death domain of Fas appears to be a necessary step (Kischkel, et al., 1995). The end result of this pathway is cell death by a distinctive mechanism characterized by nuclear and cytoplasmic condensation and DNA fragmentation.

DEPU:
Chinnaiyan A M, O'Rourke K, Tewari M, Dixit V M. (1995) FADD, a novel death domain-containing protein, interacts with the death domain of Fas and initiates apoptosis. Cell 81: 505-512

ORPL:
Chinnaiyan et al., "FADD, a Novel Death Domain-Containing Protein, Interacts with the Death Domain of Fas and Initiates Apoptosis," 81: 505-512 (1995).

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L4: Entry 1 of 41 File: USPT Jun 26, 2001

DOCUMENT-IDENTIFIER: US 6251433 B1
TITLE: Polycationic polymers

DEPR:
(i) a polynucleotide encoding a pro-apoptotic agent, including for example, fas, fas ligand, fadd, fap-1, tradd, faf, rip, reaper, apoptin, interleukin-2 converting enzyme;